Type: Poster Presentation

Final Abstract Number: 41.020
Session: Poster Session I
Date: Thursday, March 3, 2016
Time: 12:45-14:15
Room: Hall 3 (Posters & Exhibition)

Differential dendritic cells responses to infection with various serotypes of shigella

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Background: Dendritic cells (DC) are key regulators of immune response with the ability to affect both the innate and adaptive immune responses and are abundant in the gut mucosa. The severity of shigellosis varies with the serotype involved with *S. dysenteriae* (SD) producing the severest infections and complications with *S. sonnei* (SS) being at other end of spectrum usually causing mild self-limiting diarrhea. While shigellae are known to induce the apoptosis of mature DCs, there is no information in cytokine milieu of DCs incubated with different serotypes of *Shigella*e.

Methods & Materials: Monocyte derived dendritic cells (MoDCs) were developed from healthy human PBMC after 8 days of culture. They were characterized by four-color flow cytometry technique using Becton Dickinson FACS ARIA III, equipped with 488 nm and 630 nm argon laser and analysed by FACS Diva 6.1.2 Software on the basis of CD11c positive, HLA-DR positive and CD3 negative. DCs were infected with different Shigella serotypes. After 24 hour post infection, relative expression of cytokines IL-1β, IL-6, IL-8, TNF-α, IL-12p70, IL-17, IL-22 and IL-23 was studied by Real Time PCR and data was analysed by Graphpad prism 5.

Results: IL-8, IL-17A, IL-22A and IL-23 expressions were highest in MoDCs stimulated with *S. dysenteriae* serotype1 and significant serotypic differences were noted between SD & SF and between SD & SS. The transcription levels of IL-23 were down regulated in *S. flexneri* & *S. sonnei* in comparison to normal MoDCs. IL-8 appears to be a major molecule orchestrating mucosal inflammation in shigellosis. It is the primary cytokine which induces neutrophil chemotaxis. SD1 induces more Th17 response which displays pro-inflammatory functions. IL23 is responsible for the expansion of Th17 previously differentiated. IL-23 promotes the development and expansion of activated CD4+ T cells.

Conclusion: DCs are critical sentinel cells that relay microbial presence either directly or indirectly to naive T cells. In this study we found that *Shigella dysenteriae* caused maximum release of IL-8. Similarly SD also caused highest release of IL-17A and IL-22A. It was the only serotype which increased IL-23. These findings could explain more severity of SD as compared to SF and SS.

http://dx.doi.org/10.1016/j.ijid.2016.02.217

Type: Poster Presentation

Final Abstract Number: 41.021
Session: Poster Session I
Date: Thursday, March 3, 2016
Time: 12:45-14:15
Room: Hall 3 (Posters & Exhibition)

Genome wide host gene expression analysis in chicken lungs infected with avian influenza viruses

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Background: The molecular pathogenesis of avian influenza viruses vary greatly with individual bird species and virus strain. The molecular pathogenesis of the highly pathogenic avian influenza virus (HPAIV) or the low pathogenic avian influenza virus (LPAIV) in avian species remains poorly understood.

Methods & Materials: Thus, global immune response of chickens infected with HPAI H5N1 (A/duck/India/02CA10/2011) and LPAI H9N2 (A/duck/India/249800/2010) viruses was studied using microarray to identify crucial host genetic components responsive to these infection.

Results: HPAI H5N1 virus induced excessive mRNA expression of type I IFNs (IFNA and IFNG), cytokines (IL1B, IL18, IL22, IL13, and IL12B), chemokines (CCL4, CCL19, CCL10, and CX3CL1) and IFN stimulated genes (OASL, MX1, RSAD2, IFITM5, IFIT5, GBP 1, and EIF2AK) in lung tissues. This dysregulation of host innate antiviral genes may be the critical determinant of the severity and the outcome of the influenza infection in chickens. In contrast, the expression levels of most of these genes remained unchanged in the lungs of LPAI H9N2 virus infected chickens.

Conclusion: This study indicated the relationship between host antiviral genes and their roles in pathogenesis of HPAIV infection in chickens.

http://dx.doi.org/10.1016/j.ijid.2016.02.218

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Final Abstract Number: 41.022
Session: Poster Session I
Date: Thursday, March 3, 2016
Time: 12:45-14:15
Room: Hall 3 (Posters & Exhibition)

Murine model of tuberculous meningitis: New insight into understanding pathological complications of the disease

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Background: Tuberculous meningitis is most severe presentation of tuberculosis that causes mortality in one-third of the affected patients. Though an age old disease; it still remains an inefficiently treated infection of human brain. Main reason behind is